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Sleep, executive functioning and behaviour in children and adolescents with type 1 diabetes

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ABSTRACT

Objective: The aim of the study was to examine sleep, neurocognitive and behavioural functioning in children and adolescents with type 1 diabetes (T1D) compared to controls and to test whether sleep quality mediates the relationship between diabetes and neurocognitive and behavioural deficits.
Methods: Participants include 49 children and adolescents with T1D (recruited from a hospital clinic) and 36 healthy controls (age range = 6–16 years). Parents completed a survey consisting of the Sleep Disturbances Scale for Children, the Behavior Rating Inventory of Executive Functions, and the Behavior Assessment System for Children-2. Diabetic and demographic parameters were collated from medical records. The survey was posted to participants.
Results: Children with T1D compared to controls reported a higher frequency of sleep problems, and mild deficits in executive and behavioural functioning. Mediational analyses revealed that sleep quality fully mediated metacognitive functioning, externalised problematic behaviour, and internalised problematic behaviour, but not behavioural regulation.
Conclusions: Rather than the direct impact of T1D on daytime functioning, it is the consequent impact of T1D on sleep and the resulting sleep disruption which can explain much of the neurocognitive and behavioural deficits reported in children with T1D. Maintaining good nocturnal glycaemic control may play a much larger role than previously thought in regulating daytime functioning in children with T1D.

1. Introduction

Type 1 diabetes (T1D) is an autoimmune disorder characterised by the progressive loss of pancreatic islet β-cells resulting in a loss of insulin production [1,2]. T1D is one of the most common chronic diseases of childhood years. In Australian children the incidence rate has increased to 21 per 100,000 person years [3]. Children with T1D are reported to have reduced neurocognitive performance (eg, executive functioning, sustained attention, psychomotor speed, learning and memory – and as a consequence – reduced intellectual and academic performance [4–8]) and a higher frequency of problematic behaviours (eg, depression, somatisation and social withdrawal [9–14]). The neurocognitive and behavioural deficits in children with T1D have been attributed to poor glycaemic control [12,15]. It is unclear, however, as to what degree sleep disruption modulates these effects. In otherwise healthy children without impaired glycaemic control, sleep disruption is associated with reduced cognitive performance and increased problematic behaviour [16–22]. Our group and others have also shown that sleep quality mediates daytime behaviour and neurocognitive functioning in children with a range of medical conditions, for example, upper airway obstruction and eczema [18,23–27]. Taken together, these findings raise the possibility that sleep disruption may also contribute to the daytime deficits reported in children with T1D.

The literature reporting sleep data in children with T1D is limited. In a review of the literature using 'children', 'sleep', 'type 1 diabetes' and other variants as search keywords using Google Scholar, PubMed and PsychLit databases, we identified 12 studies that report objective sleep data. These include four polysomnographic [28–31], seven questionnaire (including interview) [32–36], and one combined questionnaire, polysomnographic, and actigraphic study [37] (see Table 1). To date, sleep questionnaire data have been

Abbreviations: BASC-2, Behaviour Assessment System for Children-2; BMI, body mass index; BRIEF, Behavior Rating Inventory of Executive Function; CI, confidence interval; ns, non-significant; SDSC, Sleep Disturbance Scale for Children; SES, socioeconomic status; T1D, type 1 diabetes.

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Table 1
Summary of sleep studies in children with type 1 diabetes (T1D).

Author and Date	Location and Population	Numbers (Mean Age & Gender)	Sleep Measures	T1D versus Controls	Correlational and Other Results
Blanz et al. (1993) [32]	Germany Hospital Clinic	93 T1D (18.1y, 39F) 93 Control (18.6y, 39F)	Semistructured Interview	Children with T1D compared to controls: ↑ Disturbed sleep	Diabetic severity was not predictive of disturbed sleep
Estrada et al. (2012) [38]	USA Diabetes Registry	78 T1D (5-11y = 25/78; 12–19y = 44/78 and >20y = 9/78: 41/78F) 245 Control (Relatives without T1D) (5–11y = 39/245; 12–19y = 48/245 & > 20y = 158/245: 143/245F)	Questionnaire	Both children and adolescents with T1D and relatives without T1D had similar sleep duration, sleep insufficiency, and daytime napping	Diabetes not predictive of sleep duration or daytime napping
Happe et al. (2005) [33]	Germany Hospital Clinic	46 T1D (12.0y, 25F) 50 Control (12.3y, 29F)	Questionnaire	Children with T1D and Controls had similar frequency of Restless Leg Syndrome, sleep initiation, and maintenance problems and daytime tiredness	Poor sleep initiation was associated with elevated HbA1c levels
Matyka et al. (2000) [28]	USA Hospital Clinic	14 T1D (9.4y, 5F) 15 Control (9.2y, 5F)	Polysomnography	Children with T1D compared to controls: ↑ Awakenings	The frequency of hypoglycaemia was higher in S4 compared to REM sleep
Monaghan et al. (2012) [39]	USA Hospital Clinic	24 T1D (4.1y, 12F)	Questionnaire	In children with T1D compared to general population norms: ↑ Stress in the lead up to and at bedtime ↑ Frequency parents called to bedroom after child settled ↑ Difficulty falling asleep ↑ Wake after sleep onset ↑ Slept in parental bed	Parents with 'insomniac' and 'sleep resistant' children reported higher stress and depression scores 'Insomnia' more frequent and parents more stressed leading up to or at child's bedtime in children on intensive/multiple daily insulin compared to conventional regimes
Perfect (2014) [35]	USA Hospital Clinic	50 T1D (13.4y, 21F)	Questionnaire Sleep Diary	Not applicable	Delayed bedtime on school and non-school nights associated with lower grade point average. Delayed bedtime on non-school nights associated with lower reading, mathematic and writing scores. Longer total sleep time on school nights associated with lower writing scores
Perfect et al. (2012) [37][a]	USA Hospital Clinic	40 T1D (13.5y, 16F) 40 Control (13.5y, 16F)	Questionnaire Actigraphy Polysomnography	In children with T1D compared to controls: ↑ N2 and ↓ N3 sleep ↑ Arousal Index ↑ Central Apnoea Index	Diabetes severity predictive of decreased N3 sleep Diabetes severity, higher HbA1c and higher Average Glucose levels were predictive of increased N2 sleep Average glucose levels were higher in children with OAHI > 1.5 compared to OAHI < 1.5 events/h
Pillar et al. (2003) [29]	Israel Hospital Clinic	15 T1D (12.6y, 8F) 15 Control (13.3y, 6F)	Polysomnography	In children with T1D (±hypoglycaemic) compared to controls: $Nonhypo\text{-}T1D_{AI} > Control_{AI} > Hypo\text{-}T1D_{AI}$ $Hypo\text{-}T1D_{\Delta power} > NonHypo\text{-}T1D_{\Delta power}$ $Hypo\text{-}T1D_{SE} > NonHypo\text{-}T1D_{SE}$ $Hypo\text{-}T1D_{S3+4\%} > NonHypo\text{-}T1D_{S3+4\%}$	Nocturnal hypoglycaemia associated with a deepening of sleep A greater number of awakenings in children with rapid compared to those with slow change in nocturnal glucose levels
Porter et al. (1996) [30]	Australia Hospital Clinic	20 T1D (12.8y, 11F)	Polysomnography	Children with compared to those without hypoglycaemia had similar sleep and arousal indices	Pre-sleep glucose levels were not predictive of subsequent hypoglycaemia during sleep
Varni et al. (2009) [36]	USA Hospital Clinic	83 T1D (12.9y, 39F) and 84 parents of children with T1D 157 Control (13.7y, 83F) 106 Children with Cancer in Treatment (8.2y, 76F)	Questionnaire	In children with T1D compared to controls: ↑ Sleep/Rest Fatigue scores (on both child and parental report) In children with T1D compared to children with cancer: Equivalent Sleep/Rest Fatigue scores (on both child and parental report)	On both child and parental report, higher levels of sleep/rest fatigue were associated with worse emotional, social and school functioning, psychosocial health and physical health
Villa et al. (2000) [31]	Italy Hospital Clinic	25 T1D (7.7y, 6F) 20 Control (8.8y, 5F)	Polysomnography	In children with T1D (especially in patients with poor glycaemic control) compared to controls: ↑ Central apnoea events ↑ Total-apnoeic events ↑ Duration of total-apnoeic events	Both higher HbA1c levels and longer duration of diabetes were predictive of increased frequency of Total-apnoea and Central apnoea events Central apnoea events were more frequent in REM compared to NREM sleep
Yeshayahu & Mahmud (2010) [34]	Canada Not reported	75 T1D (16.0y, 33F) 54 Control (16.3y, 31F)	Questionnaire	In children with T1D compared to controls: ↑ Sleep durations (weekday)	Insulin regime was not predictive of sleep duration[b]

[a] Subset of full diabetic cohort who underwent polysomnography. [b] Insulin regime consisted of continuous subcutaneous infusion, multiple injection or three daily injections. Hypo = hypoglycaemic, AI = Arousal Index, SDB = sleep disordered breathing, NonHypo = non-hypoglycaemic, REM = Rapid eye movement, NREM = Non-rapid eye movement, OAHI=Obstructive Apnea-Hypopnea Index, Δ power = Delta power and SE = sleep efficiency.

collected in 480, polysomnographic data in 114, and actigraphic data in 40 children with T1D. In general, the questionnaire findings indicate that children with T1D have more disturbed but longer sleep [32,34,39], however this has not been observed in all studies, Happe et al. [33] reports one group having no sleep differences compared to non-diabetic siblings/relatives [33] and the second compared to independent controls. Consistent with the questionnaire data all the polysomnographic studies reported that children with T1D report a greater number of nocturnal arousals compared to controls [28,29,31,37]. Polysomnographic findings also indicate a higher frequency of central apnoea and a trend toward lighter sleep (more N2 and less N3 stage sleep) [31,37]. The frequency of restless legs syndrome on self-report has also been examined with Happe et al. noting that it was similar to that of controls [33]. In summary, the findings in children with TID indicate that sleep problems are more frequent (eg, more nocturnal restlessness, longer periods of nocturnal wakefulness, longer and more frequent episodes of central apnoea, an increased prevalence of sleep disordered breathing and, paradoxically, a longer time spent sleeping), and are especially evident in children with poor glycaemic control [29,31,32,37,40].

To date, Perfect and colleagues are the only group that have examined the relationship between sleep and daytime functioning in children with T1D [35,37]. Perfect's group report that poor sleep habits were associated with an increased frequency of behavioural problems, reduced quality of life, higher diabetes-related worry, higher depression, and greater daytime sleepiness. They also report an association between sleep disruption and lower academic grades.

Perfect's findings raise the question as to the mechanism underlying the association between sleep disruption and impaired daytime function. A possible explanation may be the impact of sleep disruption on executive functioning. Sleep disruption is associated with impaired executive functioning in otherwise healthy children [41,42] and this effect is potentially amplified by the added impaired glucose control in children with T1D. In addition to general daytime performance, of note is that optimum executive functioning is an important factor in a child's ability to also effectively plan and manage diabetes.

Therefore, the aims of the present study were to investigate sleep and daytime functioning in children with T1D compared to non-diabetic controls and to test whether sleep significantly contributes to the relationship between diabetes and daytime functioning. Based on the findings outlined above, it is hypothesised that both nocturnal hypoglycaemic and hyperglycaemia will result in sleep disruption, thereby impairing executive function and daytime behaviour in children with T1D.

2. Method

2.1. Participants and procedure

Children and adolescents with T1D aged between 6 and 16 years were consecutively recruited over 4 months from the patient list of children attending the paediatric diabetes clinic of the Women's and Children's Hospital which is the tertiary referral centre for children in the state of South Australia and services a population base of 1.1 million. This was a sample of convenience and included approximately 20% of the diabetic clinic population. The minimum age was set at 6 years to enable assessment of executive function while the maximum age was set at 16 years to enable the recruitment of sufficient numbers for mediational analyses. Healthy controls were recruited through parents of children with diabetes who were requested to nominate a healthy non-diabetic friend of their child who might participate in the study. Children with other medical conditions or on psychotropic medication known to affect either sleep or neurocognitive functioning were excluded from the study. Eighty-five eligible children agreed to enter the study (T1D = 49 and controls = 36). All participants were Caucasian. Socioeconomic status (SES) was determined by post-code using the Australian Bureau of Statistics Index of Relative Socio-economic Advantage/Disadvantage 2011 National Census Data (national population mean = 1000 and standard deviation (SD) = 100) [43]. An SES > 1000 is indicative of increased income and occupational skills and/or training within the geographical area of residence. The study was approved by the University of South Australia and the Child, Youth and Women's Health Service Human Research Ethics Committees; all participants provided written informed consent.

An information pack with questionnaires was distributed to parents on the clinic list either while they were attending the diabetes clinic or if requested by mail. Parents of children with T1D either directly mailed or handed on the information pack to parents of controls. Parents completed all questionnaires.

2.2. Apparatus

2.2.1. Sleep disturbance scale for children

The Sleep Disturbance Scale for Children (SDSC) was used to assess sleep in the previous 6 months. The SDSC generates six scale scores: Disorders of Sleep Breathing (eg, frequency of snoring and apnoea), Initiating And Maintaining Sleep (eg, prolonged sleep onset and night awakening), Arousal (eg, sleepwalking and nightmares), Sleep–Wake transition (eg, restless legs and bruxism), Excessive Daytime Somnolence (eg, morning and day time sleepiness) and Sleep Hyperhydrosis (eg, night sweating) and a composite Total Sleep Disturbance score [44]. The SDSC yields T-scores with a mean (SD) = 50 [10]. T-scores >70 are suggestive of clinical significance. The SDSC is reported to have good construct validity and test–retest reliability [45].

2.2.2. Behavior rating inventory of executive function

The parent form of the Behavior Rating Inventory of Executive Function (BRIEF) was used to assess executive functioning in the previous 6 months; that is, the ability to selectively attend to, work with, and plan for specific information [46]. The BRIEF generates eight clinical scales: Inhibit (inhibitory control), Shift (capacity to move freely from one task to another), Emotional Control (capacity to control emotional responses), Initiate (ability to independently generate ideas and begin task), Working Memory (ability to hold information in the mind while completing a task), Plan/Organise (capacity to manage present and future task), Organisation of Materials (orderliness of work, storages and play areas), and Monitor (work checking habits and behavioural monitoring). These clinical scales combine to generate two composite indices: Behavioural Regulation (Inhibit, Shift and Emotional Control) and Metacognition (Initiate, Working Memory, Plan/Organise, Organisation of Materials and Monitor), and a composite Global Executive index. The BRIEF yields T-scores normed for age and gender with a mean (SD) of 50 [10]. A T-score >70 is indicative of clinical level deficits in executive functioning. The BRIEF is reported to have high internal consistency, good convergent validity and good test–retest reliability [47–49].

2.2.3. Behaviour and emotional functioning

The child and adolescent Parent Rating Scales of the Behaviour Assessment System for Children-2 (BASC-2) were used to assess behaviour and emotional functioning in the preceding 6 months [50].

The BASC-2 generates nine subscales: Aggression, Anxiety, Attention Problems, Atypicality (tendency to behave in ways that are immature or considered odd), Conduct Problems, Depression, Hyperactivity, Somatisation, and Withdrawal which are combined to generate two composite indices Externalising Problems and Internalising Problems and a composite Total Behaviour Symptoms Index. The BASC-2 yields T-scores normed for age and gender with a mean (SD) of 50 [10]. A T-Score >70 is indicative of clinically significant emotional and behavioural problems. The BASC-2 is reported to have good validity and reliability [51,52].

2.2.4. Diabetes

Patient medical records were used to obtain: age of diabetes onset, weight, height, body mass index (BMI) (adjusted for age and gender [53]), daily insulin regimen (multiple daily injection or continuous subcutaneous insulin infusion), HbA1c (assessed using Vantage analyser (Siemens Diagnostics, Camberley UK)), the presence over the previous 3 months of moderate hypoglycaemia (blood glucose <3.5 mM on home testing with symptoms requiring assistance from another individual) or severe hypoglycaemia (blood glucose <3.5 mM on home testing with convulsion or loss of consciousness in association), and diabetic ketoacidosis (hyperglycaemia and ketosis with serum bicarbonate <15 mM and pH < 7.3). All cases of T1D were confirmed by detection of islet antibodies (islet cell antibodies, anti-GAD antibodies or anti IA2 antibodies).

2.2.5. Statistical analysis

All data were screened for errors and normality and analysed using the Statistical Package for Social Sciences (SPSS) software program for Windows (version 20.0). *F*-tests were used to test for group differences in continuous and chi-squared tests in categorical variables. Pearson-*r* correlations were used to examine the relationship between variables.

Bootstrap mediation analyses using bootstrap conditional effects procedures [35] were undertaken to examine the effect of diabetes (not present = 2, present = 1) on Behavioural Regulation, Meta-Cognition, Externalising Problems, and Internalising Problems and whether sleep (Total Sleep Disturbance) mediated the effect of diabetes on behaviour and executive function [54] (not present = 2, present = 1). As the current gold standard for mediational analyses [35], this statistical method was chosen for the analyses as it reduces the likelihood of type 1 error while testing for mediators and covariates without relying on the assumptions of normal sampling distributions [55–58]. The statistical model for the mediational analyses is illustrated in Fig. 1.

3. Results

3.1. Demographics

Compared to controls, children with T1D had comparable age and gender distributions but significantly lower levels of maternal education and SES (Table 2). Given the significant group difference in SES, the latter was entered as an additional independent variable in the analyses investigating the effect of diabetic status on SDSC, BRIEF and BASC-2 scores. Participants were dichotomised according to the mean SES into Low and High SES groupings.

3.2. Sleep disturbance scale for children

Children with T1D compared to controls had significantly higher Disorders of Excessive Somnolence, Disorders of Initiating and Maintaining Sleep, Disorders of Sleep–Wake Transition and Total Sleep Disturbance scores (Table 3). SDSC scale scores did not significantly differ with SES level and no significant interactions between diabetic status and SES were observed.

3.3. Behavior rating inventory of executive function

Although mean scores were in the normal range, executive functioning was reduced in children with T1D compared to controls. A significantly higher frequency of executive deficits were observed in children with T1D on the Shift and Emotional Control subscales and the composite Behavioural Regulation Index and Global Executive scale scores (Table 4). Inspection of the percentage of cases in the normal (T-score < 60), borderline (T-score 60–69), and

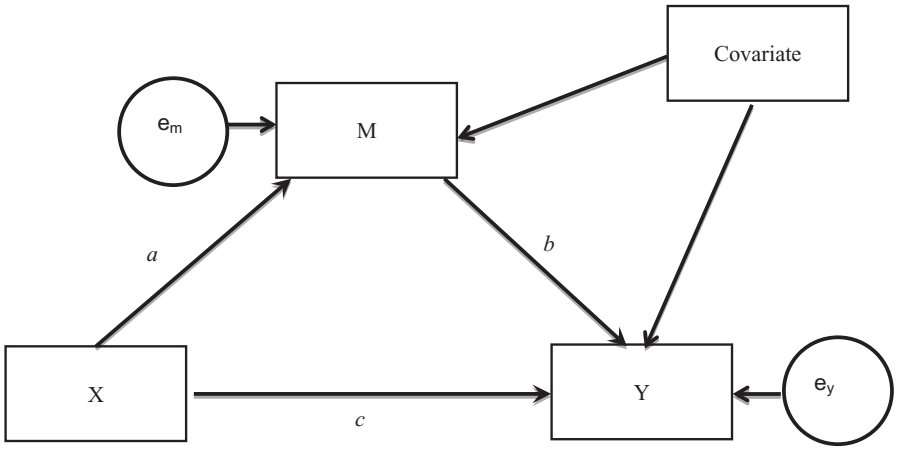

Fig. 1. Statistical model for the mediational analyses. NB: M = mediator (SDSC Total Sleep Problem Score); X = diabetic status (yes/no); Y = dependent variable (BASC-2 Internalising Behaviour and Externalising Behaviour scores; and BRIEF Behavioural Regulation Index and Metacognition Index scores); e_m = error mediator; e_y = error dependent variable; Covariate = SES; Direct effect of X on M = a; direct effect of M on Y = b; and direct effect of X on Y = c; total indirect effect of X on Y through M = $\sum$(ab).

Table 2
Demographic values for children with type 1 diabetes mellitus (T1D) and controls together with *F*-test/chi-squared results and effect size (partial eta squared η_p^2).

Demographic	Control ($n = 36$)	T1DM ($n = 49$)	F-Test /Chi-squared	Effect Size (η_p^2)
Demographics				
Gender (male) n (%)	22 (61%)	24 (49%)	0.3	
Age (years)	11.2 (2.8)	12.0 (2.8)	1.6	0.02
Mother's Highest Education Level n (%)				
Tertiary	24 (67%)	17 (35%)	8.5*	
High school	10 (27%)	26 (53%)		
Not reported	2 (6%)	6 (12%)		
SES	1044 (63)	975 (67)	22.6****	0.21
Type 1 Diabetes Profile				
BMI percentile (%)[a]	63.4 (32.3)[b]	70.2 (25.7)	0.9	0.01
HbA1c	na	8.9 (1.5)		
Duration (years)	na	4.4 (3.6)		
Diabetic Ketoacidosis (Yes %)	na	3 (6%)		
Insulin Regime				
Injection	na	28 (58%)		
Pump	na	21 (42%)		
Hypoglycaemic episodes in the last 3 months (Yes%)	na	13 (27%)[c]		

*Denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. Small effect size (η_p^2) = 0.01, medium = 0.06 and large = 0.14. NA = not applicable. [a]Gender and age adjusted. [b]N = 23. [c]N = 48.

clinical (T-score > 70) ranges revealed that a higher percentage of children with T1D on all composite test scores were also in the borderline/clinically significant range. The percentage of children with T1D in the normal/borderline/clinical range for the Metacognition Index was 67%, 20%, and 12% versus controls 81%, 14%, and 6%; for the Behavioural Regulation Index 67%, 22% and 10% versus controls 83%, 17% and 0%; and for Global Executive scale 67%, 22% and 10% versus controls 83%, 14%, and 3%, respectively.

BRIEF scores did not significantly differ with SES. A significant diabetic status by SES interactions was observed for Plan/Organise and Monitor subscale scores: where post-hoc analyses revealed in both cases that $T1D_{Low\ SES} < Control_{Low\ SES}$, $p < 0.05$.

Table 3
Mean (SD) Sleep Disturbance Scale (SDSC) for Children T-scores for children with Type 1 Diabetes Mellitus (T1D) and controls together with Two-Way ANOVA (Diabetic Status and SES) and effect size (partial eta squared η_p^2) results. Significant results are bolded.

SDSC (subscales are indented)	Control		T1D		Diabetic Status (η_p^2)	SES (η_p^2)	Diabetic Status x SES (η_p^2)
	Low SES ($n = 9$)	High SES ($n = 27$)	Low SES ($n = 33$)	High SES ($n = 16$)			
Total Sleep Disturbance Score	50.9 (6.7)	41.5 (14.2)	64.0 (16.2)	61.3 (10.6)	**23.1 (0.22)****[a]	3.1 (0.04)	1.0 (0.01)
Disorders of Initiating and Maintaining Sleep	56.4 (8.0)	54.5 (9.2)	66.4 (16.3)	72.9 (13.7)	**19.3 (0.19)****[b]	0.5 (0.01)	1.7 (0.02)
Sleep Disordered Breathing	48.9 (3.7)	49.7 (5.1)	52.0 (8.9)	50.6 (7.9)	1.2 (0.02)	0.0 (0.00)	0.4 (0.01)
Disorders of Arousal	59.8 (14.8)	50.8 (8.4)	54.8 (10.4)	53.0 (14.2)	0.3 (0.00)	3.9 (0.05)	1.7 (0.02)
Disorders of Sleep-Wake Transition	44.8 (5.9)	50.7 (9.4)	57.8 (15.7)	55.9 (14.1)	**8.2 (0.09)***[c]	0.4 (0.00)	1.5 (0.02)
Disorders of Excessive Somnolence	47.3 (6.1)	48.8 (6.5)	61.5 (14.1)	61.1 (13.6)	**22.3 (0.22)****[d]	0.0 (0.00)	0.1 (0.00)
Sleep Hyperhydrosis	48.3 (6.8)	46.3 (3.1)	52.5 (12.5)	49.5 (7.1)	2.9 (0.03)	1.3 (0.01)	0.0 (0.00)

Higher T-scores indicate a higher frequency of sleep problems. *Denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. Small effect size (η_p^2) = 0.01, medium = 0.06 and large = 0.14. SES = socioeconomic status. Mean (SD) Control vs. T1D values collapsed across SES: [a]Control = 43.8 (13.3) < T1D = 63.1 (14.6); [b]Control = 55.0 (8.9) < T1D = 68.5 (15.6); [c]Control = 49.2 (9.0) < T1D = 57.1 (15.1); and [d]Control = 48.5 (6.4) < T1D = 61.4 (13.8).

Table 4
Mean (SD) Behavior Rating Inventory of Executive Function scores (BRIEF) for children with Type 1 Diabetes Mellitus (T1D) and controls together with Two-Way ANOVA (Diabetic Status and SES) and effect size (partial eta squared η_p^2) results. Significant results are bolded.

BRIEF Variables (subscales are indented)	Control		T1D		Diabetic Status (η_p^2)	SES (η_p^2)	Diabetic Status x SES (η_p^2)
	Low SES ($n = 9$)	High SES ($n = 27$)	Low SES ($n = 33$)	High SES ($n = 16$)			
Global Executive Index	46.1 (8.9)	50.2 (9.97)	56.6 (12.7)	51.1 (9.6)	**4.4 (0.07)***[a]	0.1 (0.00)	3.1 (0.04)
Behavioural Regulation Index	46.0 (7.7)	48.4 (9.0)	56.1 (13.3)	53.3 (11.5)	**7.3 (0.08)****[b]	0.0 (0.00)	0.3 (0.01)
Inhibit	47.1 (6.7)	48.6 (8.1)	53.6 (12.5)	48.0 (9.8)	1.3 (0.02)	0.7 (0.01)	2.0 (0.02)
Shift	46.8 (8.9)	48.0 (9.5)	56.6 (12.9)	55.0 (12.8)	**8.8 (0.10)***[c]	0.0 (0.00)	0.2 (0.00)
Emotional Control	45.7 (8.3)	49.4 (10.4)	58.0 (13.0)	55.4 (13.0)	**10.0 (0.11)***[d]	0.0 (0.00)	1.2 (0.01)
Metacognition Index	46.8 (9.2)	51.0 (10.3)	55.7 (12.2)	49.6 (9.2)	2.0 (0.02)	0.0 (0.00)	3.7 (0.04)
Initiate	48.1 (10.6)	50.3 (10.6)	55.1 (10.0)	50.1 (8.2)	1.9 (0.02)	0.0 (0.00)	2.2 (0.03)
Working Memory	47.9 (7.9)	50.2 (9.8)	57.9 (13.1)	51.8 (12.2)	3.5 (0.04)	0.3 (0.00)	2.7 (0.03)
Plan/Organise	46.5 (9.7)	51.5 (11.1)	56.2 (12.1)	47.9 (9.1)	1.3 (0.02)	0.4 (0.00)	**6.1 (0.07)***
Organisation of Materials	52.3 (7.4)	51.7 (10.1)	52.9 (11.7)	51.5 (10.7)	0.0 (0.00)	0.1 (0.00)	0.0 (0.00)
Monitor	42.4 (8.7)	50.6 (13.2)	51.9 (11.1)	47.6 (9.6)	1.3 (0.2)	0.5 (0.01)	**5.0 (0.06)***

Higher T-scores indicate more impaired executive functioning. *Denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. Small effect size (η_p^2) = 0.01, medium = 0.06 and large = 0.14. SES = socioeconomic status. Mean (SD) Control vs. T1D values collapsed across SES: [a]Control = 49.2 (9.7) < T1D = 55.8 (12.0); [b]Control = 47.8 (8.7) < T1D = 55.2 (12.7); [c]Control = 47.7 (9.2) < T1D = 56.1 (12.7); and [d]Control = 48.5 (9.9) < T1D = 57.1 (12.9).

Table 5
Mean (SD) Behaviour Assessment System for Children–2 scores (BASC-2) for children with Type 1 Diabetes Mellitus (T1D) and controls together with Two-Way ANOVA (Diabetic Status and SES) and effect size (partial eta squared η_p^2) results. Significant results are bolded.

BASC-2 Variables (subscales are indented)	Control		T1D		Diabetic Status (η_p^2)	SES (η_p^2)	Diabetic Status x SES (η_p^2)
	Low SES ($n = 8$)	High SES ($n = 27$)	Low SES ($n = 32$)	High SES ($n = 15$)			
Behaviour Symptoms Index	45.9 (8.0)	44.3 (6.4)	51.7 (9.7)	48.8 (7.3)	**6.3 (0.07)*a**	1.2 (0.02)	0.1 (0.00)
Atypicality	42.5 (1.9)	44.0 (4.6)	51.7 (12.6)	50.3 (9.3)	**10.7 (0.12)***b**	0.0 (0.00)	0.4 (0.00)
Withdrawal	48.0 (9.7)	46.2 (6.9)	52.1 (9.3)	52.6 (9.6)	**5.7 (0.07)*c**	0.1 (0.00)	0.3 (0.00)
Attention	56.9 (9.0)	51.2 (5.8)	54.2 (6.8)	51.5 (8.0)	0.0 (0.00)	**5.6 (0.07)*g**	1.0 (0.01)
Internalising	48.3 (14.0)	41.3 (7.8)	54.7 (13.7)	50.3 (10.2)	**6.9 (0.08)**d**	3.7 (0.05)	1.8 (0.02)
Anxiety	54.6 (19.5)	42.4 (9.5)	52.2 (12.8)	47.8 (9.5)	0.2 (0.00)	**7.4 (0.08)**h**	1.6 (0.02)
Depression	45.8 (12.8)	43.6 (5.6)	53.4 (12.6)	50.1 (9.2)	**7.3 (0.08)**e**	1.1 (0.02)	0.0 (0.00)
Somatisation	44.9 (7.1)	43.2 (8.0)	55.9 (14.6)	53.5 (10.2)	**13.6 (0.15)*f**	0.5 (0.00)	0.0 (0.00)
Externalising	43.5 (6.9)	44.8 (7.1)	48.3 (9.6)	44.7 (7.4)	1.2 (0.02)	0.4 (0.00)	1.4 (0.02)
Hyperactivity	42.3 (5.7)	44.7 (7.8)	48.0 (10.8)	43.3 (6.4)	0.0 (0.00)	0.6 (0.01)	2.8 (0.03)
Aggression	45.0 (6.9)	45.4 (6.0)	48.4 (7.2)	46.7 (6.9)	1.8 (0.02)	0.2 (0.00)	0.4 (0.00)
Conduct Disorder	44.8 (7.7)	46.0 (7.5)	48.8 (10.3)	45.9 (9.0)	0.8 (0.01)	0.2 (0.00)	1.0 (0.01)

Higher T-scores indicate more impaired behavioural functioning. *Denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. Small effect size (η_p^2) = 0.01, medium = 0.06 and large = 0.14. SES = socioeconomic status. Mean (SD) Control vs. T1D values collapsed across SES: [a]Control = 44.6 (6.7) < T1D = 50.8 (9.1); [b]Control = 43.7 (4.2) < T1D = 51.3 (11.6); [c]Control = 46.6 (7.4) < T1D = 52.3 (9.3); [d]Control = 42.9 (9.8) < T1D = 53.3 (12.7); [e]Control = 44.1 (7.6) < T1D = 52.3 (11.6); and [f]Control = 43.6 (7.7) < T1D = 55.2 (13.3). Mean (SD) High vs. Low SES values collapsed across diabetic status: [g]High SES = 51.3 (6.5) < Low SES = 54.7 (7.2); and [h]High SES = 44.3 (9.7) < Low SES = 52.7 (14.1).

3.4. Behaviour and emotional functioning

Despite having mean scores in the normal range, children with T1D compared to controls had a higher frequency of problematic behaviours. In particular, they reported significantly higher Atypicality, Depression, Somatisation and Withdrawal subscale scores and the composite Internalising and Behaviour Symptoms Index scores (Table 5). Although means were in the normal range, a higher percentage of children with T1D compared to controls had composite scores in the borderline/clinically significant range. Inspection of the percentage of cases in the normal (T-score <60), borderline (T-score 60–69) and clinical (T-score > 70) ranges for Externalising Problems in children with T1D was 87%, 11%, and 2% and for controls 97%, 3%, and 0%, respectively. The percentages for Internalising Problems in children with T1D were 72%, 15%, and 13% and controls was 97%, 0% and 3%, respectively. The percentages for Total Behaviour Symptoms Index in children with T1D was 72%, 15%, and 13% and controls were 97%, 0%, and 3%, respectively. Children with a lower compared to higher SES also had significantly higher Anxiety and Attention subscale scores. No significant diabetic status by SES interactions was observed.

3.5. Diabetic severity

To test for the effect of hypoglycaemia severity on key composite sleep, executive functioning and behavioural variables, we divided the children with T1D into those who reported either no ($n = 35$) or moderate hypoglycaemic episodes in the previous 3 months ($n = 12$) (one child with severe hypoglycaemia was excluded from the analyses). *F*-test analyses revealed no significant group differences (all $p > 0.05$). Additional *F*-test analyses were also undertaken to examine differences in children with T1D on continuous subcutaneous insulin infusion ($n = 21$) versus multiple daily injection ($n = 28$). The latter analyses revealed no significant group differences on any composite sleep, executive functioning or behavioural score (all $p > 0.05$).

3.6. Correlational analyses

In children with T1D, a strong relationship was observed between impaired sleep (Total Sleep Disturbance score) and both reduced executive functioning (Metacognition and Global Executive scores) and higher problematic behaviour (Externalising Behaviours, Internalising Behaviours and Behaviour Symptom Index) (Table 6). Although not reported, we also explored the relationship between HbA1c levels and the composite sleep, executive functioning and behavioural scores, but no significant correlations were observed (all $r(45) < 0.25$, *ns*).

3.7. Mediational analyses

As SES was shown to differ between participant groups, it was entered in all mediation analyses as a covariate. As per convention for mediational analyses, the covariate results are not reported.

3.7.1. Externalising behaviour

The results from this analysis confirmed a mediational model with the presence of diabetes indirectly influencing externalising behaviour through its effects on sleep. As can be seen in Fig. 2a and Table 7, children with diabetes experienced sleep difficulties ($a = -17.5$), and in turn, children with sleep difficulties demonstrated greater levels of externalising behaviour ($b = 0.2$). A bias-corrected bootstrap confidence internal for the indirect effect ($ab = -4.21$) based on 5000 bootstrap samples was entirely below zero (−7.86 to −1.45). There was no evidence that the presence of diabetes influenced externalising behaviour independent of its effects on sleep difficulties ($c' = 1.7$, $p = 0.4$).

3.7.2. Internalising behaviour

The results from this analysis confirmed a mediational model with the presence of diabetes indirectly influencing internalising

Table 6
Correlation values for the relationship between Sleep Disturbance Scale for Children (SDSC) Total Sleep Disturbance T-score and key composite behaviour and executive functioning scores.

Key Neurocognitive and Behaviour T-scores	SDSC Total T-score[a]
Behavior Rating Inventory of Executive Function	
Global Executive	0.45**** (0.12/0.53****)
Behavioural Regulation Index	0.33*** (0.10/0.25)
Metacognition Index	0.44**** (0.13/0.60****)
Behaviour Assessment System for Children–2	
Behaviour Symptoms Index	0.57**** (0.15/0.64****)
Internalising Behaviours	0.58**** (0.22/0.58****)
Externalising Behaviours	0.42**** (0.15/0.53****)

*Denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. [a]Pearson-*r* values reported for the combined sample and separately in parenthesis for controls and children with T1D.

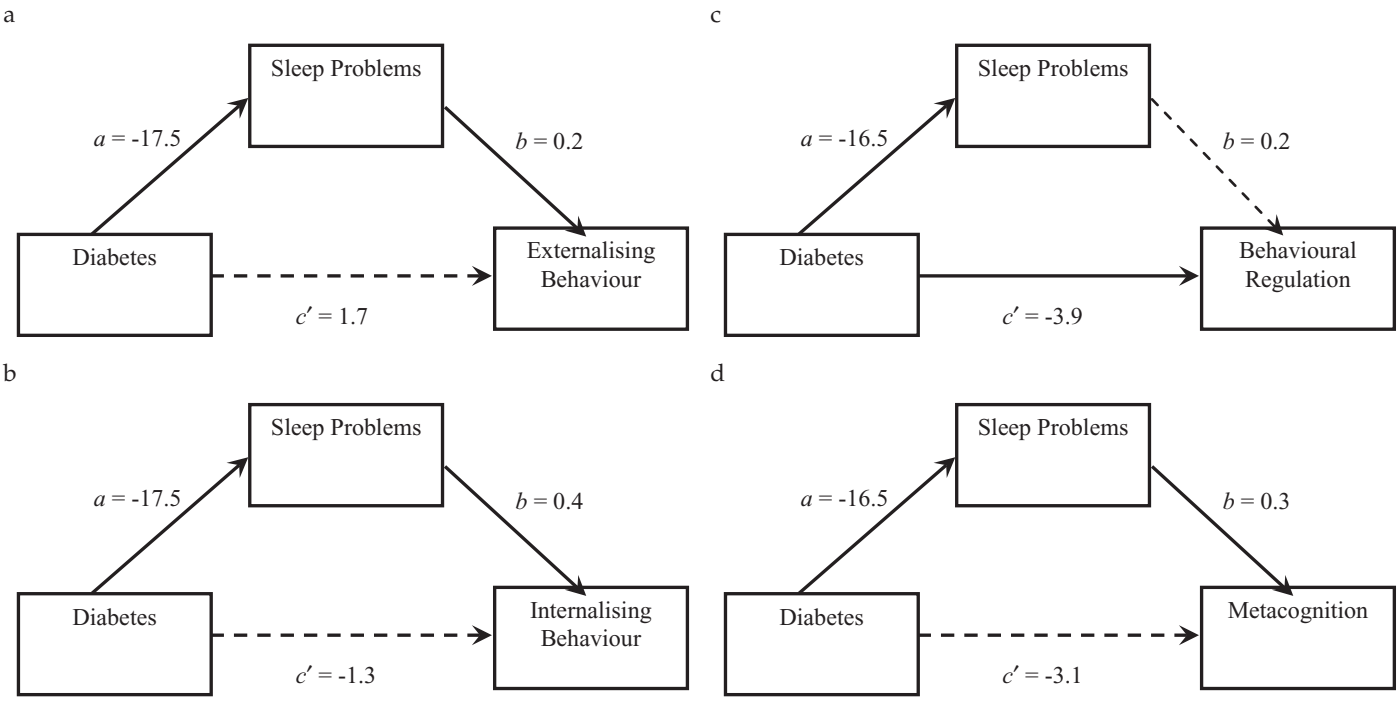

Fig. 2. All numbers represent unstandardised coefficients. (a) Sleep problems mediate the effects of the presence of diabetes on externalising behaviours. (b) Sleep problems mediate the effects of the presence of diabetes on internalising behaviours. (c) Sleep problems do not mediate the effects of the presence of diabetes on behavioural regulation. (d) Sleep problems mediate the effects of the presence of diabetes on metacognition.

behaviour through its effects on sleep. As can be seen in Fig. 2b and Table 7, children with diabetes experienced sleep difficulties ($a = -17.5$), and in turn, children with sleep difficulties had higher levels of internalising behaviours ($b = 0.4$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab = -6.3$) based on 5000 bootstrap resamples was entirely below zero (−10.27 to −3.19). There was no evidence that the presence of diabetes influenced internalising behaviours independent of its effect on sleep difficulties ($c' = -1.3$, $p = 0.7$).

Table 7
Model coefficients for the effect of diabetes on behaviour (BASC-2) and executive function (BRIEF) through sleep, controlling for socioeconomic status.

Antecedent		Consequent						
		M (Sleep)				*Y*		
		Coeff.	*SE*	*p*		Coeff.	*SE*	*p*
						BASC-2 Externalising Behaviour		
X (Diabetes)	*a*	−17.5	3.5	<0.001	*c′*	1.7	2.2	0.435
M (Sleep)		—	—	—	*b*	0.2	0.1	<0.001
Constant		113.0	21.9	<0.001		24.6	13.9	0.081
		$R^2 = 0.35$				$R^2 = 0.18$		
		$F(2, 79) = 21.4$, $p < 0.001$				$F(3, 78) = 5.9$, $p = 0.001$		
						BASC-2 Internalising Behaviour		
X (Diabetes)	*a*	−17.5	3.5	<0.001	*c′*	−1.3	3.0	0.672
M (Sleep)		—	—	—	*b*	0.4	0.1	<0.001
Constant		113.0	21.9	<0.001		59.4	18.7	0.002
		$R^2 = 0.35$				$R^2 = 0.37$		
		$F(2, 79) = 21.4$, $p < 0.001$				$F(3, 78) = 15.0$, $p < 0.001$		
						BRIEF – Behavioural Regulation		
X (Diabetes)	*a*	−16.5	3.4	<0.001	*c′*	−3.9	3.1	0.218
M (Sleep)		—	—	—	*b*	0.2	0.1	0.078
Constant		118.4	21.6	<0.001		55.7	20.1	0.007
		$R^2 = 0.34$				$R^2 = 0.14$		
		$F(2, 82) = 21.4$, $p < 0.001$				$F(3, 81) = 4.3$, $p = 0.008$		
						BRIEF – Metacognition		
X (Diabetes)	*a*	−16.5	3.4	<0.001	*c′*	3.1	2.8	0.279
M (Sleep)		—	—	—	*b*	0.3	0.1	<0.001
Constant		118.4	21.6	<0.001		37.8	18.3	0.042
		$R^2 = 0.34$				$R^2 = 0.21$		
		$F(2, 82) = 21.4$, $p < 0.001$				$F(3, 81) = 7.1$, $p < 0.001$		

3.7.3. Behavioural regulation

The results from this analysis did not reveal mediation (see Fig. 2c and Table 7). Although children with diabetes experienced sleep difficulties ($a = -16.5$), sleep difficulties did not significantly influence behavioural regulation ($b = 0.2$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab = -2.59$) based on 5000 bootstrap resamples contained zero (−7.03 to 0.95), indicating there was no significant difference between the direct effect and total effect of diabetes on behavioural regulation. Although the indirect effect did not reach significance, it is worth noting that there was no evidence that the presence of diabetes influenced behavioural regulation independent of its effect on sleep difficulties ($c' = -3.9$, $p = 0.2$). This final result indicates that at least part of the variance in behavioural regulation may be due to sleep difficulties.

3.7.4. Metacognition

Finally, the results from this analysis confirmed a mediation model with the presence of diabetes indirectly influencing metacognition through its effects on sleep. As can be seen in Fig. 2d and Table 7, children with diabetes experienced impaired sleep quality ($a = -16.5$), and in turn, children with sleep difficulties demonstrated greater impairments in metacognition ($b = 0.3$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab = -5.43$) based on 5000 bootstrap resamples was entirely below zero (−9.35 to −2.13). There was no evidence that the presence of diabetes influenced metacognition independent of its effects on sleep difficulties ($c' = 3.1$, $p = 0.3$).

4. Discussion

In this study, children with T1D compared to controls had reduced executive functioning, a higher frequency of problematic behaviours and a higher frequency of sleep problems. The novelty of the current study is that it examined whether sleep itself mediated the relationship between TID, executive functioning, and daytime behaviour and found that it did. These findings highlight the importance of the clinical assessment of sleep problems in children during routine diabetic clinic visits.

Children with T1D had reduced daytime functioning compared to healthy controls. Specifically, they had reduced mental flexibility and ability to modulate emotions leading to poor behavioural regulation and a higher frequency of depression and withdrawal resulting in a higher frequency of internalised problematic behaviours. They also had elevated somatisation scores, but it is unclear whether this is explained by increased stress or the diabetes itself. However, not all behavioural domains were impaired with no deficits observed in externalised problematic behaviour and apart from a mild deficit in working memory there were no deficits in metacognition (ie, cognitive self-awareness and regulation). An examination of the domains impacted by T1D suggests that emotional control was more adversely affected than executive functioning. Children with T1D also had a higher frequency of sleep problems, especially with sleep initiation and maintenance, sleep–wake transition and daytime sleepiness. The deficits observed in executive functioning and behaviour and, similarly, sleep are consistent with findings elsewhere in the paediatric diabetic literature [13,33,40,59,60].

The brain regions integral to executive functioning and behavioural regulation, that is, pre-frontal cortex and limbic system, contain a high density of insulin receptors and are thought therefore to be sensitive to abnormal blood glucose levels [61]. Notably, the functions of these brain regions are also sensitive to sleep disturbance, the consequences of which in non-diabetic children are a range of neurocognitive and behavioural deficits [16,41,62–64]. In children with T1D, poor glycaemic control is associated with both reduced executive functioning and problematic behaviour and, importantly, with poor sleep [4,7,33,61]. Thus, neurocognitive and behavioural functioning in diabetic child is potentially susceptible to a double-risk profile of abnormal blood glucose control and poor sleep.

In the present study, the contribution of sleep difficulties to daytime functioning was explored using mediation analyses. Sleep difficulties were shown to fully mediate externalised behaviour, internalised behaviour and metacognition (Figs 1, 2a, b and d), but not behavioural regulation (Fig. 2c). Taken together, the mediational results suggest that sleep difficulties rather than diabetes per se are a stronger predictor of daytime functioning in children with T1D. To date, only Perfect and colleagues have examined the relationship between sleep and daytime functioning in children T1D [35,37]. In the first of a brace of studies, they report that longer time spent in N2 sleep was associated with a lower grade point average, a higher frequency of emotional/behavioural/learning problems and higher depression scores [37]. In the second study, they reported that delayed bedtimes on non-school nights were associated with lower mathematics, reading, and writing grade point averages [35]. Perfect's findings suggest that sleep problems impact on academic progress in children with T1D, while the present findings suggest that this may be explained by the impact of disturbed sleep on executive functioning, which itself is well known to impact academic progress [65–67].

Consistent with the previous results, the questionnaire findings in the present study revealed a higher frequency of problems with sleep initiation, maintenance and transition and greater daytime sleepiness in children with T1D. We did not observe a higher frequency of sleep disordered breathing on parental report.

Based on adult findings we would predict a role for both nocturnal hypoglycaemia and conversely hyperglycaemia in sleep disruption [68]. Three of the studies identified in our review examined the association between hypoglycaemia and sleep and found that hypoglycaemia was associated with a greater number of central apnoeas, increased slow wave sleep activity and also longer sleep and hence greater sleep efficiency [28,29,31]. By contrast, one group found that sleep and arousal indices were comparable in children with hypoglycaemia compared to those without hypoglycaemia [30]. The rate at which nocturnal glucose levels fluctuate may also be important with one group reporting that the more rapid the change the higher the frequency of awakening [29]. In contradistinction to hypoglycaemia, hyperglycaemia (eg, elevated HbA1c levels and higher mean blood glucose levels) in children with T1D has been associated with poor sleep initiation, reduced slow wave sleep activity, increased N2 sleep, decreased N3 sleep, increased frequency of obstructive events, and longer self-reported sleep time [33,35,37]. In the present study, we also examined the relationship between HbA1c levels and problematic sleep but failed to find any significant associations.

This study is not without limitations. The evaluation of both sleep quality and daytime functioning was reliant on parental self-report with its potential biases (eg, mono-informant bias or common rater effect, and using the same informant to determine both sleep and behaviour problems which may inflate correlations due to item overlap). Another aspect is that parents of old compared to younger children may be less familiar with their child's sleep habits, thereby affecting reliability of sleep estimates. In addition, nocturnal blood glucose levels were not monitored. Nonetheless, we did compare children with T1D who reported moderate hypoglycaemic episodes (ie, requiring intervention from another person), including nocturnal hypoglycaemic episodes, in the previous 3 months to those who did not, which revealed no significant differences in the composite executive functioning, behaviour and sleep scores. In future studies, the addition of polysomnography, formal neurocognitive testing rather than parental report, the inclusion of teacher evaluations and continuous 24-h interstitial glucose monitoring will help further elucidate the role of sleep in daytime functioning in children with T1D. A further limitation was that those in the control group were from a slightly higher SES with a higher percentage of mothers with a tertiary education. The addition of SES into the current analyses revealed that children with low compared to high SES were more likely to report problems with attention, somatisation and anxiety. Although low SES is known to be a risk factor for both poor sleep and problematic behaviour [69,70], the inclusion of SES as a covariate in the mediation analyses did not significantly impact the relationships that were tested between diabetes and daytime functioning.

In conclusion, clinicians need to be aware that normal sleep architecture is important for daytime functioning, and its disruption is a significant predictor of neurocognitive and behavioural deficits in children with T1D. The optimisation of executive functioning in children with TID by the timely recognition and treatment of sleep problems may help affected children better manage their glucose control. Therefore, the inclusion of a sleep history during regular clinical reviews may help identify children at risk of neurocognitive and behavioural deficits. The present results will help in the development of evidence-based guidelines for sleep hygiene as part of overall diabetic education and clinical care.

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Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.08.011.

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